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FUSIFORM RUST OF SOUTHERN PINES— A CRITICAL REVIEW

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Fusiform Rust of Southern Pines — A Critical Review

Felix J. Czabator

This paper attempts a comprehensive summary of present knowledge of fusiform rust. It comprises an exhaustive survey of the literature, and also notes major current research. In addition, it includes references to and comparisons with other plant rusts. The subject is far from closed: there are many admissions of ignorance and suggestions for investigation. I hope the review will facilitate research that will lead to eventual control of the disease.

THE DISEASE—OCCURRENCE, DISTRIBUTION, AND ECONOMIC IMPLICATIONS

The fusiform rust of southern pines, caused by Cronartium fusiforme Hedgc. & Hunt ex Cumm., is widely distributed from the eastern shore of Maryland to Florida, Texas, and southeast Arkansas (298, 351). The disease is a major obstacle to the management of slash and loblolly pine plantations (3, 104, 125, 126, 143, 299, 300, 351, 360), and may cause catastrophic losses of nursery seedlings of these species unless intensive control measures are taken. The disease has not been reported from other countries, although species of Cronartium with oaks as alternate hosts are found in Canada, Mexico, Honduras, and Guatemala.

Cronartium fusiforme is heteroecious, with one part of the life cycle on the leaves of oaks and the alternate generation on pine stems and branches. The oak hosts comprise more than 20 species. The effect of the fungus on the oaks is slight, though when infection is abundant the premature shedding of diseased leaves causes some reduction in growth (135).

While the most frequent pine hosts are loblolly pine (Pinus taeda L.) slash pine (P. elliottii var. elliottii Engelm.), longleaf pine (P. palustris Mill.) and pond pine (P. serotina Michx.), 23 other species and varieties of native and exotic pines also may be infected (135, 170, 268, 302). Shortleaf pine (P. echinata Mill.) is conceded to be practically immune (140, 169, 298), although Wakeley says that localized infections of shortleaf pine occur in western North and South Carolina (363, p. 92).

On pine hosts, the disease is manifest by a spindle-shaped swelling or gall that develops at or near the point of infection. On nursery seedlings, the gall usually is within 1 to 4 inches of the groundline. The galls may or may not have a witches'-broom of adventitious branches arising from the swollen tissue (31, 60, 134, 139, 298).

Infection occurs through cotyledons, needles, tender stem tissue, and occasionally graft callus (45, 171, 298). The first signs of infection, purple to blue-purple spots or blotches, occur in 4 to 10 weeks after inoculation (171); the spots later become yellow-green (135). When older trees have one or more stem galls, it is a safe assumption that infection occurred on the branches and the fungus grew through the branch to the stem. Growth of the branch galls generally is somewhat faster toward than away from the stem (120, 298). Galls more than 15 inches from the stem usually kill the branch before the fungus can invade the stem (120, 208, 296, 299).

Galled seedlings usually die within 3 or 4 years after infection, when the cambium surrounding the gall is killed. On older trees, the gall usually is restricted to less than half of the circumference of the bole. Frequently the cambium is killed (298, 363) and the gall becomes a canker, an open wound subject to attack by wood-decaying fungi and insects (135, 136, 239, 386). In established plantings, mortality ascribable to fusiform rust may be four to nine times that from other causes. Occasionally seedlings recover from infection

(311); in these cases, the gall tissue becomes discolored and is overgrown by vigorous healthy host tissue. Recovery from the disease may be purely a host reaction, or may be the result of temperature extremes (27, 355).

The gall tissue generally has lower specific gravity than normal wood, and is somewhat weaker (54, 239). Although the increased diameter of the stem at a gall frequently compensates for the weakness of the wood, stem breakage by wind is a major cause of loss in trees of pulpwood size or larger (194, 386).

Sections of trees with galls or cankers are unacceptable for either lumber or pulpwood, and must be culled. Young trees with numerous stem and branch cankers are so deformed as to be worthless even for pulpwood (299). The growth rate of infected trees is reduced both by the demands of the parasite and the diversion of wood into multiple branches rather than stem tissue.

Mortality is more frequent in slash pine (194, 195, 225, 238, 299, 363) than in other species. Loblolly pine is more prone to a slowing in growth as a result of multiple stem cankers and witches'-brooms. In slash pine, and to a lesser extent in loblolly, a butt and root rot caused by **Polyporus tomentosus** var. **circinatus** (Fr.) Sartory & Maire is associated with basal galls and cankers (33, 277), and is another source of windthrow and loss.

Pines are susceptible from the cotyledonary stage until they become 50 years or more in age (171, 360, 363), but infections decrease as tree ages increase (103, 104, 299) and progress of the disease is slowed as trees gain size (298). The greatest mortality and deformation are in plantations up to 5 years of age (6, 141, 142, 289, 295, 298, 301, 363). Infection in the latter part of a rotation causes very little mortality and little loss of wood through defect (385).

Within the botanical range of **C. fusiforme**, incidence on the susceptible pines varies from occasional to very high; the most severe infections

Left: Typical fusiform rust galls on branches of slash pine.
Right: Telia of **Cronartium fusiforme** *on the under side of a water oak leaf.*

occur in the southern part of Mississippi and Alabama, and in southeast Louisiana. The zone of moderate to severe infection (10 to 30 percent) generally coincides with the natural range of slash pine.

The coincidence of severe infection with the natural range of slash pine suggests the possibility of mathematically evaluating the effect of climatic conditions on the potential severity of the disease. Hocker (151, 152) and Bethune (25), using multiple regression techniques, determined the climatic factors that limit the natural range of loblolly and slash pine. A similar procedure may be practicable to define the range and local severity zones of fusiform rust.

A systematic survey to delimit the present range and establish severity zones is sorely needed. The last published survey was that made by Lamb and Sleeth (195) in 1938 and 1939. Subsequent reports of distribution, range, and severity (3, 340, 363) are based only on occasional observations by foresters and pathologists.

In spite of the lack of systematic data, there is no doubt but that the incidence of rust is increasing (336, 361). The paucity of early references (11, 230, 334, 335) to observations and collections on pines indicates that the disease was relatively rare prior to 1900. By 1947, Siggers and Lindgren (299) commented that "15 years ago trunk and branch cankers on southern pines were of interest mostly to classifiers of tree diseases," and observed that fusiform rust had become "increasingly prevalent in the Gulf South in the past 35 years." They cited instances of some stands in which 90 percent of the trees had one or more stem cankers. In 1929, Hayes and Wakeley (129) reported that fusiform rust was of minor importance in plantations near Bogalusa, Louisiana. Today, incidence is very high in this same general area. In other parts of the South, severe mortality caused by rust, with 60 to 80 percent of the survivors having trunk infection, is not rare in slash pine plantations still below minimum pulpwood size (351, 363).

The 1938-1939 survey classed central Louisiana and Florida as areas of light infection; in 1959, the writer examined a 3,000-acre slash pine plantation near Alexandria, Louisiana, in which 70 percent of the trees had multiple stem and branch cankers.

Weber (378) reported infection of 64 percent of the trees in a stand near Gainesville, Florida; and several plantations near Foley, Florida, have more than 40 percent of the stems infected.

The mere percentage of stems infected, high as it may be, is not the best measure of the disease's economic impact. The reduction in stand productivity that results when growing space is occupied by deformed, dwarfed trees must be considered, as well as actual volume losses caused by death or windthrow of the infected trees (19, 298, 386).

The same increase in infection is evident in nurseries. The presence of rust in nurseries was first recognized about 1937 (194, 299, 329), and losses of 15 to 35 percent were reported for some nurseries in 1938 and 1939 (302). Control measures were initiated at the Ashe Nursery at Brooklyn, Mississippi, in 1939, and by 1941 spraying with bordeaux mixture or ferbam was standard practice in southern nurseries. In 1949, however, infection exceeded 65 percent in some nurseries in spite of routine protective sprays, and losses approached 60 percent in 1957. Part of these sporadically high losses may be attributed to careless spraying, but undoubtedly the greater part resulted from increased inoculum potential, since at one nursery 35 percent infection occurred in 1959, even after a recommended spraying program had been put into effect (95, 96, 297).

Much of the increase is attributed to off-site planting of the host pines, i.e., to the use of susceptible loblolly and slash pine on typical longleaf pine sites and to the planting of slash pine outside its natural range (104, 137, 194, 351, 363). Off-site planting does not account for the severe infection in nurseries, however. Except for a temporary (and unsuccessful) oak-eradication program near some nurseries (194, 297, 302, 363), there has been no serious attempt to reduce spore production by elimination of infected hosts (74, 194, 299).

Genetic susceptibility of the hosts may have a significant effect on incidence (20, 140, 169, 314, 363), and genetic variation (physiologic races) may exist in the rust itself (363).

Phenological variations in both the oak and pine hosts account for some local increase of the rust (30, 102, 104, 291, 292, 293, 294, 363). These variations frequently occur after late winter fires or cultivation of pine plantations (30, 102, 293, 294, 388).

Nearly all of the earlier research on fusiform rust has been on chemical control of the disease in nurseries. This emphasis has been both feasible and

practical. The heavy concentration of plants and high values in a nursery justify intensive control. The average southern nursery may produce, per acre, one million trees valued at $5,000 (363). Reducing infection from an average of 30 percent to 10 percent thus would represent a saving of at least $1,000 per acre, considerably more than the cost of control measures now practiced.

Though at present considered uneconomical (299, 300, 351, 363), control in plantations and natural stands is a very important area for future research. Anticipated planting in the South for the two decades 1955-1974 is estimated at approximately 515,000 acres per year (337), and control in these plantations would have far-reaching effects. In addition, every effort should be made to reduce the losses in natural stands. The southern pines are very important to the national economy, accounting for 28 percent of the total annual cut.

THE FUNGUS

The rusts are obligate parasites of higher plants, worldwide in distribution, and cause more economic loss than any other group of fungi which infect plants. Wherever higher plants exist, some rusts occur (11).

The rusts are of unusual scientific interest because of their many spore forms (up to five distinct stages), and most species have a striking change of hosts. The heteroecious rusts, particularly, have an exceptionally high degree of specialization apparently associated with critical nutritional demands. It has been postulated that the rusts obtain from one host certain nutrients for spore development but that establishment of the fungus requires another nutritional complex obtainable only from the alternate hosts (11). The nutrition factor may be part of the mechanism of host specificity (susceptibility-resistance).

Taxonomy and Nomenclature

The genus **Cronartium** is separated from other rusts by the presence of sessile, one-celled teliospores that are united in columns; a prominent aecial periderm; and pedicillate urediospores. The genus is heteroecious, with pycnia and aecia being produced on the plants of the genus **Pinus**, and the uredia and telia on leaves of various herbaceous and woody dicotyledonous plants (12).

For North America, eight to 15 species of **Cronartium** are recognized, according to various interpretations and authorities (12, 31, 57, 58, 76, 88, 135). Several differ so little in taxonomic characteristics that some classifiers consider them as one species, in spite of extreme differences in gross morphological effects on the aecial hosts. (Taxonomic criteria in the rusts are based on the microscopic characters of the spore stages on the host that bears the perfect, or telial stage.) On the other hand, some conifer rusts are classed as species of **Cronartium** by inference, although the telial stage is not known. Strictly speaking, these must be considered species of the form-genus **Peridermium**, the aecial stage of rusts on conifers, with peridia and catenulate aeciospores (255). The inference that species of **Peridermium** are only incompletely known species of Cronartium is probably sound, in that all of the previously established species of Peridermium were shown to be Cronartium when the complete life cycle was determined. It is also possible that some species, as the Woodgate rust (411), may have lost the uredinial-telial stages.

Within the geographic range in which the fusiform rust occurs, several species of **Cronartium** are known:

Cronartium quercuum (Berk.) Miyabe ex Shirai (=**C. cerebrum** Pk.?)
C. comandrae Pk.
C. fusiforme Hedgc. & Hunt ex Cumm.
C. strobilinum Hedgc. & Hahn

Of these species only **C. comandrae** and **C. strobilinum** can be easily distinguished. **C. comandrae** has very distinctive aeciospores, and the telial stage is found on **Comandra umbellata** (L.) Nutt. **C. strobilinum** bears aecia only on the cones of slash and longleaf pine, and both the uredial and telial stages on oak leaves are reported to be very distinctive (135).

Verrall (353) found in east Texas a gall-forming pine-oak rust that differs from **C. quercuum** and **C. fusiforme** in physical appearance and phenology on pine, although the uredia and telia resemble those of **C. fusiforme**.

The pine-oak rusts have been the subject of considerable debate as to whether they are one species, varieties of one species, or three species (9, 10, 133, 255, 377, 381). Arthur (12, 13, 15) classed all of the pine-oak rusts, including **C. strobilinum**, as Cronartium quercuum (Berk.) Miyabe ex Shirai. Hedgcock and Siggers, in disagreement with this classification, considered the four American species, **C. cerebrum**, **C. fusiforme**, **C. conigenum** (Pat.) Hedgc. & Hunt, and **C. strobilinum**

to be distinct from each other and from C. quercuum (135). This is a change from the earlier conclusion of Hedgcock and Long that C. fusiforme and C. cerebrum are identical, and not even sufficiently differentiated to constitute separate races (134). Spaulding (317) commented that C. quercuum and C. cerebrum have been accepted as identical until recently when significant differences were found. He does not cite any references, and may have been commenting on the publication by Hedgcock and Siggers (135).

Nomenclature in the rusts was confused and ambiguous until 1950, when the adoption of Article 69 of the International Code of Botanical Nomenclature established the basis for naming the rust fungi (197). By this ruling, the name of the perfect (telial) stage is set up without reference to any corresponding epithet for an earlier imperfect stage. Utilizing the earlier publication of Hedgcock and Hunt (268), Cummins (57) legalized the combination Cronartium fusiforme by a Latin description and references to the type specimen.

The synonomy of this rust is:

Cronartium fusiforme Hedgc. & Hunt ex Cumm.
Mycologia 48: 603. 1956.

Cronartium fusiforme (Peck) Hedgc. & Hunt
Phytopathology 8: 316. 1918.
(Nomen nudum)

Cronartium cerebrum (Peck) Hedgc. & Long
J. Agr. Res. 2: 247. 1914.
(Pro parte; Nomen nudum)

Peridermium fusiforme A&K
Bull. Torrey Bot. Club 33: 421. 1906.

Cronartium quercuum (Berk.) Miyabe ex Shirai
Bot. Mag. 13: 74. 1899.
(Pro parte)

Peridermium cerebrum Peck
Bull. Buffalo Natur. Sci. 1: 68. 1873.

Hedgcock and Hunt cited **Peridermium fusiforme** Peck in synonomy when they established the new combination C. fusiforme (268). This obviously was a lapse, since there is no evidence that Peck ever used the specific epithet P. fusiforme. By 1914, Hedgcock and Long formed the combination Cronartium cerebrum by using Peck's P. cerebrum as a base (134, 248), and later, in 1918, Hedgcock and Hunt noted Arthur and Kern's P. fusiforme as a species distinct from Peck's P. cerebrum (133). However, in 1914, Arthur and Kern had cited their P. fusiforme (14) as a synonym of P. cerebrum Peck, thus tacitly acknowledging these to be the same species (15).

Apparently Arthur (12) was on firm ground in relegating C. fusiforme, C. strobilinum, and C. cerebrum to synonomy with C. quercuum. The type specimen of C. quercuum is American, which makes C. cerebrum a synonym (13). Since C. cerebrum was the base of C. fusiforme, legally any of the pine-oak rusts must be referred to C. quercuum regardless of the pine host or geographical range.

Several alternate possibilities may be developed.

1. The pine-oak gall rusts are all one species, and the gall shape results from the host-reaction only, possibly as an expression of host physiology or genetics. Evidence for this conjecture is the consistent occurrence of only globose galls on shortleaf pine.
2. The rusts are varieties or races of the same species. Both globose and fusiform galls are formed on one host species, loblolly pine; witches'-brooms develop very frequently on the fusiform galls but never on the globose galls (31).
3. The rusts are taxonomically and physiologically distinct species.

The techniques for testing these hypotheses are feasible but time-consuming. The first requirement is a supply of aeciospores collected in various parts of the country and authenticated as to the putative rust species. After oak hosts are successfully inoculated, the sporidia can be used to inoculate the southern pine species, in all combinations. Since multiple infections often occur on the same pine tree it would seem feasible to inoculate different branches of one host tree with various species or races of the rust, thus eliminating variations due to host reaction. After the pine hosts have been successfully inoculated, microchemical or serological tests of the gall tissue can be made to detect similarities or differences. Serological comparisons of homogenates from ungerminated aeciospores of C. fusiforme and C. quercuum are reported to be antigenically different (105). Anatomical studies and compatibility tests with the pycnial fluid may also be useful (56). Dwinell reports that black oak, Quercus velutina Lam., is hypersensitive to infection by aeciospores and urediospores of C. fusiforme and normosensitive to C. quercuum (80).

Regardless of the reaction of the aecial hosts to the disease, it is impossible to differentiate these species by reference to the legal diagnoses. Boyce (32) comments that the uredia and telia of **C. fusiforme** cannot be distinguished from those of C. cerebrum. The published descriptions (57, 135, 268) of the uredinal and telial stages are sufficiently broad and generalized to encompass the normal range of variations encountered in the field; the mean dimensions of any spore form of **C. fusiforme** fall within the published range of that form of **C. quercuum**. When the intergradation of a major population is so nearly complete that it is difficult to tell where one begins and another ends, the only practical solution is to describe the group as a single variable species.

It would appear, then, that the pine-oak gall rusts in the South probably are no more than specialized forms of C. quercuum. Both globose and fusiform galls may be found on the same tree; frequently a gall may be globose on one end and tapered at the other (255). Globose galls may elongate to a fusiform shape in 1 year. In the nursery, many rust-infected seedlings develop globose galls, although a high percentage have the typical fusiform gall (133). Hedgcock and Siggers report that inoculating pines with sporidia from authentic C. cerebrum resulted in fusiform swellings that later became spherical (135).

Even cross inoculations are not conclusive. Hedgcock and Siggers (135) report that inoculation of slash, longleaf, and loblolly pine seedlings (among others) with telia (sporidia) of **C. strobilinum** resulted in infection of these seedlings, with small galls on the slash and loblolly, and "a surprising amount of mortality."

Another interesting feature of the taxonomic problem is the possibility that spot identification based on "typical" fusiform galls may be erroneous at times. **Cronartium comandrae** has been reported recently on shortleaf pine in Missouri and Arkansas (24) and on loblolly pine in Tennessee (266). Earlier records show that this species has been collected on loblolly pine at Mt. Olive and Moss Point, Mississippi, both localities in fusiform rust territory (230). An authenticated herbarium specimen of a comandra gall on shortleaf pine was examined by the writer and appeared to be identical with a fusiform rust gall. Thus, infection by **C. comandrae** may account for the occasional report of **C. fusiforme** on shortleaf pine. Minor differences in gross appearances between these two fungi, such as the nature of the peridium, may be overlooked, especially if the peridium is ruptured.

Genetics and Variation

Although physiologic races are common in the cereal rusts (11, 12), races of Cronartium have not been conclusively demonstrated (117, 140). The wide range of host species suggests that the fungus evolved simultaneously with its hosts, and that for the most part a gene-to-gene relationship for susceptibility does not exist (249). In any event, physiologic races usually are less likely in the haploid mycelium (in pine) than in the dicaryotic form (on the oaks). Thus, there may be races with different abilities to produce telia and sporidia.

However, the first recognizable, consistent variation in the fungus was the recent discovery of white aeciospores on two galls (182). These aeciospores were used to inoculate oaks, which produced white uredia and sporidia. Subsequent inoculations of slash and loblolly pine resulted in the development of white aeciospores again (181). It is not known whether the white-spored form is a distinct race of the fungus or only a color mutation. The infection pattern (development of signs and symptoms, incubation time, gross appearance of the galls) of the white-spored form is identical with that of the normal orange-spored form. Lines developed from single uredial pustules show a diffuse pattern of segregation by the pine spore stages, with a range in color of pycnia and aecia from white through intermediate yellow to the typical orange color.

It is of interest that the autoecious **Peridermium harknessii** J. P. Moore has an albino strain that is widely distributed on ponderosa pine (229).

Life Cycle and Spore Forms

Cronartium fusiforme has five spore forms, which are produced in succession. The life cycle is complete in 2 years or more.

Infection of the pine host by sporidia from the oaks occurs in early spring, producing a septate, branched, intercellular, uninucleate mycelium characterized by wide hyphae (3.0 to 4.3 μ averaging 3.5 μ) and inflated haustoria (166, 176). This mycelium produces pycnia in extensive, shallow cavities under the epidermal layer of the galls.

At maturity, the oblong to elliptical pycniospores average 2 × 3.8 μ in size and are exuded in viscid, yellow-tinted drops according to Hedgcock and Siggers (135). Buller, however, working with specimens sent to him by Bailey Sleeth, reported that the exposed pycnidia, including masses of pycnidiospores, are bright orange-red (41). Our own observations agree with Buller's description, and chemical analyses show carotenes, which are characteristically orange-red, within the spores.

The pycnial drops appear to be attractive to many insects. Ants of several species swarm about the bases of infected seedlings in autumn and early winter and often are found on seedlings in which the swelling or gall is barely perceptible.

The function of the pycniospores is still controversial. They are not infective, since repeated attempts to inoculate the hosts with them have been unsuccessful (11, 101). Craigie (51) concluded that they function in diploidization of the rusts, a conclusion supported by Cummins (56). Craigie deduced that the mycelia which produce the pycnia are of two kinds (+ and −), each derived from a uninucleate sporidium. All of the pycniospores of a pycnium are of the same sex, and function as spermatia. Certain hyphae (flexuous hyphae) in the host tissues serve as receptive cells, and upon receiving spermatia of the opposite sex become binucleate and then initiate the dicaryotic phase. The nuclei are conjugate, however, and do not fuse until the telial stage is reached (56).

Hyphal fusion and nuclear migration between cells at the beginning of the aecial stage have been demonstrated in the majority of the rusts (11, 52, 404).

It is unlikely that pycniospores are nonfunctional. The rusts are extremely specialized and variable and unnecessary spore forms have tended to disappear. Physiologic specialization (races) of the cereal rusts is conceded to be genetically controlled, and thus sexuality is implied. Buller (41) observed that specimens of C. fusiforme and C. cerebrum from Mississippi and Florida had flexuous hyphae, similar to those of C. ribicola. He inferred that these hyphae initiate the sexual process.

Pycnia are produced 1 to 2 years after infection, and the pycnia and aecia are formed in alternating generations on the same galls, the aecia appearing the year after the pycnia (the following spring) (135). Wolf and Wolf (404) state that pycnia do not repeat themselves but occur only once in the life cycle.

Pycnial production is reported to occur from October to April (135). However, systematic observation of galls for 4 years disclosed that the pycnial ooze first appeared about mid-October, but ended either in mid-November, early December, or mid-December. Jewell et al. (176) state that pycnial sporulation occurs from late October to mid-December, and agree that aecia follow the pycnia immediately, being produced on the same gall although not in the same area of the gall. In our own laboratory, pycnia developed within 6 months after inoculation of pines with sporidia. The dates that pycnia first appear are considered to be diagnostic criteria to differentiate between C. fusiforme and C. quercuum; C. fusiforme appearing from October to April; C. quercuum from December to February (135, 379). Systematic observations revealed no definite pattern or sequency of pycnial-aecial sporulation. Pycnia were sometimes followed by aecial sporulation for 2 or 3 consecutive years, but usually aecia developed in the same area of a gall where pycnia had been produced the previous autumn.

The mechanism of pycnial and aecial sporulation should be investigated. Pine seedlings maintained in the greenhouse under a uniform temperature (70 to 85° F.) and at a constant day of 20 hours produced pycnia about the end of October, even though the plants were inoculated at different times from early December to mid-June. Thus the date of pycnial sporulation was influenced neither by the length of time the rust had been established in the pines nor by the differences between greenhouse conditions and those in nature.

Of the hundred or more species of rust known to infect pines in the genera Cronartium, Peridermium, Coleosporium, and Melampsora, the pine hosts always bear the aecial stage (11, 12, 414). In Cronartium the aecial mycelium is perennial in the pine host, and may form aeciospores from the same infection many years in succession (11, 41, 50, 66). The aeciospore initials first cut off from two to 10 apical peridial cells with rudimentary intercalary cells, forming the peridium, a tissue enclosing the sorus. Subsequent divisions of the aeciospore initials form binucleate aeciospores, each with an intercalary cell, developing into long chains of aeciospores (50, 82).

Each aecial sorus of C. fusiforme produces tremendous volumes of thick-walled orange spores that average 15.5 × 25.1 μ and range from 13-18 × 22-28 μ (135). The spores are obovate to ellipsoid, and coarsely verrucose but generally with a

smooth area at the broader end and extending up one side. A definite germination pore is not present; the germ tube emerges by dissolving or rupturing thin areas of the cell wall. Frequently multiple germ tubes emerge from different parts of the spore (368.)

The aeciospores are capable of germination as soon as they are released from the sorus. They maintain high germinative capacity for 2 months or more when stored at temperatures below 10° C. (290). Successful storage requires careful drying over calcium chloride for several days to reduce contamination and deterioration by **Penicillium** and other species of molds (4, 276, 370), and screening to remove extraneous matter, such as insect larvae. The spores maintain high viability during storage in liquid nitrogen at —196° C. (376). Loss of viability is associated with a gradual change in color from orange to gray (135, 290).

The loss of color is related to a chemical degradation of the carotenes that give the spores their orange appearance. This degradation is not the cause of lessened viability, since faded spores frequently germinate very well. Furthermore, the white-spored form had only traces of the carotenes in any of the five spores, but viability was excellent (182).

The aeciospores are formed and disseminated in early spring, from February to April (135). They apparently are food for insects (11); frequently they disappear abruptly at the close of the season, and the aecial cavities appear to have been scraped clean. Hirt (149) states that the aecial sorus of **C. ribicola** Fischer is a discrete structure, which may be removed intact before the peridium ruptures. This observation holds true for **C. fusiforme** also, and probably accounts for the sudden disappearance of the spores.

The aeciospores are carried by wind and air currents to the oaks, where they germinate on the leaves. Germination in the laboratory takes place in 2¼ to 4 hours, at an optimum temperature of 21° C. Good germination occurs over a range of temperature from 17 to 22° C; the lower and upper limits for germination are 11 and 29° C. (290). The aeciospores require only an endogenous supply of water: nutrients added to the germination medium rarely affect the process, although citric and oleic acids cause some stimulation, especially of spores after storage (368). Products of lipid metabolism, including lower-chain fatty acids, are inhibitory (365). No dormancy occurs, but aeciospores produced during prolonged cold wet weather may be nonviable.

Artificial inoculations indicate that infection occurs only on young leaves (135, 313). A decrease in susceptibility with age has also been reported for leaves of Ribes petiolare Dougl. inoculated with **C. ribicola** (261). On the other hand, leaves of **Ribes triste** Pall. do not become infected when very young, nor when hardened; infection is limited to fully expanded leaves (116).

One of the gall-forming rusts on pine, **Peridermium harknessii**, is facultatively autoecious; that is, the aeciospores are capable of reinfecting pine (226, 227, 254, 356). On Scotch pine, the Woodgate rust (207, 221, 292) apparently is also propagated by aeciospores, since the telial stage and alternate host are not known. This phenomenon has prompted attempts to show that the aeciospores of **C. fusiforme** may reinfect pine, but all trials have been unsuccessful (135).

It is entirely possible that both autoecious and heteroecious forms of **C. fusiforme** exist. Pine-to-pine infection would not be possible with spores from the heteroecious form (221). The autoecious form, if it exists, must be only a very small part of the rust population, and the probability is very great that it has not been used in inoculation trials. A common criterion for identifying autoecious forms is the absence of pycnia (226, 227, 333). However, the pycnia of **C. fusiforme** are produced 6 months or more before the aecia (135, 176), and the identification of the autoecious form would require very careful, repeated examination of many galls for several years. The attempt might be inconclusive even then, since there is also the possibility that the aecial form may be repeated for several years on the same gall, while the pycnia do not repeat themselves (404).

Germination of the aeciospores results in a binucleate mycelium in the oak leaf tissue, from which uredia and telia are produced. The mycelium in the oak leaf is massed in the mesophyll, but ramifies into the palisade layer and under the upper epidermis. The greater mass of the mycelium develops in the region of the lower epidermis, where the uredia and telia are formed. The haustoria are small in the early stages of infection, but gradually expand. Usually only one uredium, followed by one or more telia, is produced by the mycelium derived from one aeciospore infection (82).

Within about 1 week after inoculation, the hyphal tips of the rust in the oak leaf become aligned into a pseudopalisade beneath the lower

epidermis. Two or three cells are cut off from each hypha; these become the peridium. Relatively large rectangular basal cells are then formed under the peridial cells. These basal cells divide, forming an elongated binucleate cell and a residual basal cell. The elongated cell rounds off into a urediospore, and again divides to form a relatively vacant stalk cell (intercalary cell) attached to the urediospore cell. When the urediospores are mature, they detach from the stalk cell (82).

In 9 to 11 days after inoculation the uredium appears as a very small (0.14 × 0.16 mm.), pale green spot on the host leaf (135). The developing uredium ruptures the lower epidermis of the leaf, and the peridium ruptures almost immediately thereafter (82). Occasionally uredia and telia may be formed on both upper and lower leaf surfaces (173). Cummins' (57) type description of the uredia indicated sparse, minute sori with a central opening and a tenuous peridium on the lower surface of the leaf.

The urediospores are ovate to obovate, averaging 13 to 16 × 17 to 24 μ, hyaline, thick-walled (2 to 2.4 μ), echinulate (57). Hedgcock and Siggers (135) indicate average dimensions of 13.3 × 19.1 μ, with the intersextile range of 12 to 15 × 17 to 21 μ, and walls 2.0 μ thick. The presence of carotenes in droplets within the protoplasm gives the spores an orange color. The white-spored form of C. fusiforme produced colorless urediospores, with only traces of carotenes.

The urediospores are capable of reinfecting the oak host under certain conditions. Siggers reported that initial inoculation of oak leaves resulted in secondary uredia in only 33 percent of the cases and that oak inoculation could not be carried beyond the second generation in the greenhouse (135). However, in the greenhouse at Gulfport, successive urediospore inoculations have been carried on for several years at approximately 10-day intervals. On the other hand, the urediospore stage apparently is not essential in the life cycle of C. fusiforme. Aeciospore inoculation of older oak leaves results in the production of telia without intervening uredia (82, 313).

Secondary infections probably are not common in nature. Uredia are relatively rare in the field; the most common signs of oak infection are the telia. At Gulfport, hundreds of susceptible oak seedlings have been grown without infections on the same greenhouse bench with seedlings bearing uredial pustules. Urediospores begin germination in 1 to 1½ hours. The optimum temperature is 18° C., with good germination over a range of 15 to 20° C. The upper and lower thermal limits are 8 and 29° C. (290).

Uredia on oak leaves have been observed to appear and disappear in 18 days (291). The urediospores are capable of limited germination (and infection) after 223 days of storage at 4 to 10° C. (290). For some rusts the temperature at which the urediospores are produced will influence both the germinative capacity of the spores and their response to storage. With cereal rusts, urediospores formed at 16° C. had better viability and germination than those which developed at 22 or 29° C. (150).

Urediospores of selected strains of C. fusiforme, including the white-spored form, are in liquid nitrogen storage in the American Type Culture Collection.

Telia are formed from the same mycelium as the urediospores (82). Inoculations of oaks with aeciospores produce telia more frequently than uredia (135). When the supply of nutrients or condition of the host is unfavorable for production of uredia, the rust may grow directly from the aeciospore mycelium to the telial stage (10).

Hedgcock and Siggers reported that telia were produced on the lower surface of oak leaves in about 20 to 35 days after inoculation with aeciospores (135). Siggers later found that teliospores developed and germinated 8 days after inoculation of oak leaves with aeciospores, and concluded that the primary telium developed in 6 days after inoculation (290). In our own studies at Gulfport, Mississippi, the average time for telia to appear was 7 ± 1.5 days after aeciospore inoculation. Seven percent of the inoculated plants produced telia in 5 days. Urediospore inoculation produced telia in 8.5 ± 1.5 days; 8 percent of these inoculations produced telia in 6 days.

The telia develop from February to June, appearing as dark brown, cylindrical or thread like structures averaging 0.1 mm. in diameter and 3 to 5 mm. in length (57, 135). Neither color nor dimensions of the telia should be considered diagnostic features. In the greenhouse at Gulfport we have observed numerous telia more than 15 mm. long, and Hedgcock and Siggers note that length may be related to age and environment (135).

When telia follow uredia, the teliospores and telia develop from the same mycelium as the uredium; generally one to three telia develop on the circumference of the uredium. Whether telia are pro-

duced alone or follow the uredia, their early stage is similar to that of the uredium: a pseudopalisade is formed, with basal cells. After the peridial cells are formed successive divisions of the hyphal tips form chains of teliospores only; there are no intercalary (stalk) cells (82).

Telia normally form on the lower surfaces of oak leaves, but occasionally are amphiphyllous (173), and a telium has been found on young stem tissue (83).

From 10 to 50 or more chains of teliospores appear simultaneously, each chain becoming cemented to its neighbors by a material, called the matrix, that probably is an end product of the process by which the teliospore reaches quick maturity. The average daily growth of a telium is about 100 μ (290). Since the spores are produced by divisions of the teliospore initials at the base of each chain, those at the tip of the column are more mature than those at the base (82, 135), and germinate earlier.

Siggers reported that the teliospores are not viable if separated from the telium matrix (290), but he admits that the techniques used for separation — boiling the telial column in 4-percent hydrochloric acid, or nitric acid — are not likely to maintain viability. Both Siggers (260) and Pierson (290) state that the matrix is pectin, a conclusion supported by our own tests. Microchemical tests (114) for various plant substances were negative for all but pectin, but digestion with a wide range of pectinase solutions for up to 24 hours failed to affect the telium matrix. Digestion for longer periods of time resulted in bacterial decomposition of the telium.

The matrix is considerably harder than the teliospore walls. Attempts at mechanical separation only rupture the spores. Yet the delicate probasidia from spores interior to the column readily make their way through the matrix to the outside.

Teliospores are single-celled, thin-walled, oblong to oblong-fusiform; dimensions are 12 to 16 $\times$ 30 to 44 μ (57) or 10 to 20 $\times$ 25 to 54 μ (135). Their color is masked by the dark matrix. Chemical analyses are inconclusive, because of the matrix, but the teliospores contain the same carotenes found in the other four spore forms, as well as unidentified alcohol-soluble pigments that probably are polyphenol compounds.

As a general rule, germination of the teliospore produces a four-celled probasidium, each cell of which bears a basidiospore or sporidium on a short sterigma. Although nuclear details have not been confirmed for C. fusiforme, it is assumed that the paired nuclei fuse during maturation of the teliospore, that reduction division occurs early in the course of probasidial development, and that each of the four daughter nuclei resulting from the reduction division migrate to one of the sporidia and re-establish the uninucleate phase of the rust (50).

The teliospore of most rusts is a resting spore, but in C. fusiforme it is capable of germination soon after the telia are formed. That is, the spores apparently do not require a dormant period for maturation. The absence of a resting period for teliospores is characteristic of the microcyclic lepto forms of rust (404), but Arthur (11) observes that it is an adaptive feature, without taxonomic significance.

Teliospores of C. quercuum remained viable up to 40 days (241). Siggers (290) observed that only a few telia of C. fusiforme had viable spores after approximately 35 days' storage at temperatures up to 35° C., and concluded that most of the fungus in the leaf tissue had been killed by exposure to the heat and dryness. His tests were conducted with only segments of oak leaves, and under natural conditions on intact leaves the telia may be viable for longer periods.

Telia produced substantial numbers of sporidia 32 days after their appearance, but spore production was negligible at 46 days (267). Teliospores began to germinate in less than 1½ hours in an atmosphere of high humidity (306). Germination first occurred at the tip of the telial column and continued in a progressive manner to the base of the telium. Drying the telium soon after teliospore germination began, then incubating it again, resulted in germination of teliospores nearer the base of the telium.

The number of successive germination periods through which the telia may pass depends on the length of the telium and the length of time that high humidity prevails (306). Six to ten hours of high humidity may induce germination of only some of the teliospores, leaving the remainder capable of germination at some later date. Telia of C. fusiforme incubated at 20° C. over water agar sporulated continuously for more than 10 days. Telia which germinate under water, or under low oxygen stress, give rise to mycelial-like strands instead of probasidia (188).

Teliospores germinate well at temperatures between 17 and 23° C., and the optimum is about 21°. Siggers (290) found that germination did not

occur at temperatures below 15° or above 26° C., but Snow (306) obtained limited and delayed germination at 12° C.

The sporidia are abjected from the pro-mycelium approximately 3 hours after germination of the teliospore, according to Siggers (290), but the time required varies with environmental conditions (188). In a saturated atmosphere, sporidia have been collected 1½ hours after the telia were incubated (306).

The sporidia are considered to be very sensitive to adverse conditions, and quickly lose their germinative capacity. Sporidia which had been deposited on a glass slide became plasmolized in a few minutes after they were removed to the relatively dry atmosphere of a greenhouse (290). They begin germinating in about 2½ hours, at an optimum temperature of 22° C., although germination of 13 to 93 percent occurred at temperatures from 19 to 25° C. Germination failed at temperatures below 15 and above 26° C. (290). The optimum temperature was 20° C. (307). In the laboratory germination occurs only in water, or in a saturated atmosphere (147, 290).

Kais (180) described the sporidia as one-celled, ellipsoid to ovoid, smooth, thin-walled, apiculate, and with a granular content. The mean dimensions of normal orange sporidia are 12.8 ± 1.3 × 9.6 ± 0.8 μ, with maximum range of 9.3 to 18.6 × 7.0 to 12.8 μ. Dimensions of the white-spored form (182) are not significantly different.

Sporidia produced by germinated teliospores in a telial column. (X500)

Secondary, tertiary, and quaternary sporidia are commonly formed when primary sporidia germinate (147, 180, 188, 233), and are also capable of infecting pines (275). The successive sporidial forms may have a survival value to the fungus, since they frequently are produced and abjected forcibly when the primary sporidium germinates on plants that do not normally become infected (233).

The abjected sporidia are carried by air currents. They germinate on the surface of the pine needles (occasionally on juvenile bark), and the germ tubes enter the host through stomata or occasionally by direct penetration (16, 148, 246).

Physiology

The physiology of C. fusiforme has been studied in detail only with respect to temperature requirements for spore germination, and to sensitivity to fungicides of the spores and mycelium. Some spore-germination studies have been conducted on the host plants in the greenhouse (82, 181, 307), but most have been done in vitro. Many substrates, humidities, temperatures, and spore ages have been evaluated (135, 290). Temperature requirements for each spore form have already been discussed.

When optimum conditions for spore germination are established, these are assumed to be applicable to field conditions and are made the basis for recommendations on control of the disease (95, 96, 97, 106, 107, 292, 297, 351, 363). Occasionally however, control recommendations fail to allow for the range of favorable conditions, or for the possibility of spore germination under adverse conditions (77, 410).

Practically all investigators agree that the humidity necessary for spore germination must be close to saturation, possibly 97 percent or higher (11, 48, 147, 290, 309, 319, 344), and that urediospores and sporidia germinate best on a free water surface. Germinative capacity will vary considerably with the kind and duration of moisture conditions that the spores encountered prior to incubation (200, 241, 306, 310, 345, 346).

Germination usually is assessed in free water on a microscope slide, or in the humid atmosphere of a Van Tiegham cell (147, 290). Excellent

germination is obtained on distilled water agar (123, 233, 241, 365, 366, 368), particularly if the glassware is thoroughly cleaned with sulfuric acid-bichromate solution (211). No particular advantage is gained from various artificial media (11, 46). Use of host plant extracts in the germination substrate (11, 36, 394), has given disappointing results, but it is claimed that the age and maturity of the host tissues have a marked effect on germination (150, 290, 261, 276, 321).

Compounds other than extracts of host tissue have been reported to stimulate germination in vitro. Among these are hydrogen peroxide, sugar solutions, vitamins, and some organic acids (11, 98, 257, 303, 370, 394). However, neither vitamins nor hydrogen peroxide stimulated germination of C. fusiforme aeciospores (365). In some cases, germination of other rust spores is stimulated by fungicides in low concentrations (242).

In the laboratory, both the pH of the medium and the composition of the buffer solutions used to adjust the pH have a decided influence on spore germination. All spore forms of C. fusiforme germinate over a wider range of pH (4.5 to 7.0) in vitro than occurs in or on the host plants. Acetate and bicarbonate buffers are inhibitory to germination, even in the presence of otherwise stimulatory compounds (365).

Inhibition may be caused by environmental factors, such as light intensity (71, 84, 122, 150, 196, 224, 247), an excess of carbon dioxide, or a lack of oxygen (11). Fatty acids C_1-C_{12} inhibit aeciospore germination of C. fusiforme (365, 366, 367). Some complex organic acids, produced within the spores by the metabolism of the rust itself, act as self-inhibitors (5, 322, 327, 365, 400). Self-inhibitors of C. fusiforme aeciospores probably are volatile or water-soluable substances; attempts to germinate aeciospores in mass usually are unsuccessful, although the same spore lot will germinate well when the spores are dispersed. Gelatin granules absorb the self-inhibitor, permitting germination in mass (365). From the ease with which they can be germinated, urediospores and sporidia of C. fusiforme appear not to have a self-inhibitor.

Most inorganic salts (as those of potassium, sodium, magnesium) have no effect on spore germination, but salts of the heavy metals (mercury, copper, iron, zinc) are toxic (49, 70, 99, 157, 183, 258, 316, 373, 396). Nickel chloride is toxic even at low concentrations (93, 365). Toxicity effects probably are the result of protoplasm degradation following penetration of the spore wall by the toxic molecule. The spore walls apparently are highly permeable even to large molecules such as those of cycloheximide (365). Surfactants have been reported to damage the cytoplasmic membrane irreparably, allowing the cell contents to leak out of spores (90).

The influence of light—intensity, duration, wavelength, and alternation of light and dark periods—needs further investigation. In other rusts, even the more resistant aeciospores and urediospores are adversely affected by blue and ultraviolet light but germinate well in red or white light (71, 224). Germination of aeciospores and sporidia of C. fusiforme is almost completely suppressed by irradiation with ultraviolet light (2537A) at 70,000 ergs/cm.^{2}, though the sporidia gradually recover and produce secondary sporidia 2 or 3 days after irradiation. The aeciospores undergo bacterial decomposition without recovering. The significance of suppression and recovery of sporidia after irradiation is obscure. Although 2537A is lethal to most fungi and bacteria (189, 262), this wavelength can produce hydrogen peroxide from water. The peroxide is said to stimulate spore germination (11).

Complete darkness at the time of inoculation and in the early stages of infection reduces incidence and severity of infection by urediospores of Puccinia graminis (122). Teliospores of nearly all rust species germinate more readily if exposed for several days to alternating light and darkness, while continued darkness inhibits germination (11). After germination, establishment of the fungus (formation of appressoria and host penetration) requires light intensities of from 2,000 to 5,000 foot-candles. Above 9,000 foot-candles, the effect of light is inhibitory (84).

The coincidental loss of aeciospore viability with a change in color from bright orange to grey (135, 290) suggests a gradual loss or oxidation of a pigment system within the spore. At Gulfport, chemical analyses showed that approximately 15 percent of the dry weight of aeciospores was lipids, chiefly alpha and gamma carotenes.

Although a gradual loss of carotenes is associated with aging of the aeciospores, it does not appear that the carotenes are sources of energy for germination. High germination rates were found in some aged spore lots with very low carotene content. Aeciospores and urediospores of the white-spored form (182) showed only traces of carotene, perhaps from orange spores remaining in collecting equipment (252, 330, 331). Addition of caro-

tenes to the medium did not stimulate germination. Reduced viability of stored, faded aeciospores can be partially overcome by the addition of citric or oleic acid to the medium (365, 370).

Until recently, studies of spore germination and artificial inoculations were possible only during spring, generally from March to May. Aeciospores are produced in the early part of this range, March and April; but urediospores are usually difficult to find, and telia frequently are nonviable by early May (135, 290). Storage of the rust spores has accordingly received considerable attention. Urediospores and teliospores of the cereal rusts have been stored for up to 3 years (179). Telia of C. ribicola are viable for 3 months, and telia on leaves of Ribes have sporulated after overwintering on the ground (318).

Successful storage of any spore requires that respiration be reduced. The reduction may be accomplished by freeze-drying or by storage in oil, in inert atmospheres, or at low temperatures (44, 47, 85, 118, 121, 165, 190, 200, 212, 213, 285, 286, 325, 366). Storage at 10° C. of **C. fusiforme** aeciospores for several months, and of urediospores for 223 days, was reported by Siggers (290). However, viability declined rapidly. Aeciospores stored at 1 to 4° C. maintain some viability for more than a year, and thus continuous stocks of aeciospore inoculum are available. At about 5° C., urediospores maintain good viability for approximately 3 months. Aeciospores and urediospores of C. fusiforme can be stored in liquid nitrogen (366). Freezing, freeze-drying, or refrigerated storage in an atmosphere of nitrogen has not been successful with any of the other spore forms of **C. fusiforme.**

The precise interpretation of spore germination tests requires standardized conditions and techniques, replications to establish confidence limits, and an adequate supply of spores (217, 218, 219). Above all, the pattern of germination of each spore form must be established within the effective temperature and humidity ranges. A population of spores represents a heterogeneous mixture of individual spores that vary in maturity, dormancy, and sensitivity; and a plot of germination over time usually results in a sigmoid curve, since the variations follow a normal distribution. A dosage-response curve, converted to a straight line by the use of a logarithmic-probability grid, permits an accurate determination of average germination percent under the conditions of the test (28, 75, 228). (This is the "LD-50" of fungicide tests, and here time may be considered equivalent to a fungicide, "dosage" being the units of time required for germination.) The slope of the dosage-response curve measures the variations in environmental conditions, and the age of the spore population.

The physiology of the fungus cannot, however, be studied by extrapolating from the results of germination tests. Spore germination is a phenomenon distinct from growth or establishment of the fungus. The rusts are obligate parasites, and are capable of only limited development except in the tissues of the host plant (409).

Cultures of rusts are maintained by inoculating plants of the susceptible host, then collecting the spores and inoculating the next host plant, which is usually the alternate host unless the repeating urediospores are used (46, 376). With fusiform rust, artificial inoculation of pine seedlings with sporidia (135, 170, 171, 172) and inoculation of oaks with aeciospores and with urediospores are well-known techniques (9, 10, 132, 134, 135, 290). These inoculations usually are separate and isolated operations; straight-line inoculations, to maintain continuity of the same mycelium, have been made with the white-spored form (181). Single-spore inoculations of pine with sporidia of C. fusiforme have been made at Gulfport, but with an extremely low degree of success.

The stage of maturity of the host tissues, both pine and oak, limits establishment of the rust (171, 298, 313). A greenhouse is required to maintain stocks of host plants in a susceptible condition (86, 247, 387), particularly at times of the year when outdoor conditions are unfavorable for infection. At Gulfport we found that a day length of 18 to 20 hours, produced by a combination of incandescent and special fluorescent lights, prevents dormancy of the host plants (86, 202). Temperatures were maintained near those prevailing in the spring (21 to 29° C.). Inoculations frequently were made at night, to take advantage of more favorable temperatures, and high humidities were maintained by intermittent mists actuated by short-cycle timers (344). Plants were covered with plastic bags for 24 hours after inoculation (320). The host plants were kept vigorous by fertilization and watering.

The effectiveness of these procedures may be recognized by the fact that at Gulfport repeated uredinial inoculations have been made at 10-day intervals for more than a year; telia and sporidia can be produced on demand and on schedule for

chemical analyses or for inoculating pines. Pines have been successfully inoculated in every month of the year.

Inoculation techniques range from dusting aeciospores, urediospores, and sporidia on the host plant, to procedures such as inserting telia, or strips of agar with cast sporidia, into slits in the pine stem; injecting suspensions of sporidia into pines with a hypodermic syringe; and transferring the active mycelium from one plant to another by bark-patch grafting (46, 116, 135, 169, 171, 185, 245, 278, 344).

The technique developed by Snow (306, 307), is extremely effective. Two clear plastic boxes are connected by a short piece of tubing. Oak leaves bearing germinating telia are placed in the upper portion of one box, and the seedling to be inoculated is positioned in the other, smaller, box near the tube. A vacuum applied to the smaller box draws spore-laden air from the large box across the seedling. The number of spores per unit area of pine tissue may be adjusted by regulating the rate of airflow.

As knowledge of fungus physiology increases, it may be possible to develop procedures for maintaining C. fusiforme in artificial media. Some advances in this line have been made with the cereal rusts; structures similar to appressoria have been developed by manipulation of light and substrate (84, 163, 247, 412), and mycelium of one race of Puccinia graminis has been grown on artificial media (397, 398). Mycelium of the cedar rust has been successfully maintained in excised host-tissue cultures (59, 158, 159) and mycelium of C. ribicola on cambial explants (127) of western white pine.

Mycelium of C. fusiforme also has been maintained in slash pine tissue cultures at the Gulfport laboratory (369). Establishment of slash pine cultures has been more consistent, and with a higher percentage of success, than has been reported for other rusts. The use of infected tissue culture in a modification of the bark-patch grafting procedure (245) has possibilities for establishing clonal lines of the fungus.

The chemical composition of host tissues may limit both germination of the spores and establishment of the fungus. Aeciospores of C. fusiforme germinate equally well on susceptible or resistant oak leaves. The fungus does not become established in the older (resistant) leaves, although appressoria, the earliest infection structures, are common (135, 290, 313).

The nature of stimulation or inhibition of rusts by the host tissues has not been determined (34, 35, 38, 62, 63, 98, 119, 244). Some host plant metabolites, particularly organic acids, have a pronounced effect on in vitro germination and growth of C. fusiforme spores and hyphae (365, 370). They may stimulate or inhibit spore germination, accelerate germ tube growth and increase tube length, or cause reduced tube growth and multiple branching (368). The adaptive function of these modifications needs investigation. Accelerated growth and increased length probably are favorable for entrance and establishment. Multiple branching may interfere with penetration or may allow the fungus a greater number of entry points.

In general, the mode by which the germ tube of a rust fungus enters the host depends on the type of spore from which the hypha has arisen. Aeciospore and urediospore germ tubes generally enter through open stomata (82), while sporidia can penetrate directly (246), as well as through stomata (11, 16, 193). It is entirely possible that differences in mode of entry are strictly random. If stomatal penetration is significantly more frequent than direct penetration for some spore form, however, the tropic response of the germ tube becomes interesting. What are the factors that direct or attract the hypha to the stomate?

Variations in light intensity and moisture stress influence the stomatal mechanism of the hosts. If infection occurs primarily through open stomata, there may be times when the stomata are open for less time than is required for spore germination and penetration of the germ tube. This lack of synchronization may account for low rust infection in some years.

Direct observations have revealed that the aeciospore germ tubes may bypass several oak leaf stomata before penetrating one more distant. Therefore, for fusiform rust, germ tube length in vitro (148) may not be a valid index of capability for infection.

Establishment of the rust in the host is accomplished by the penetration of an absorbing organ, the haustorium, into a host cell (82, 269). After establishment of the fungus, a delicate balance between the rust and the host cells must be achieved. If the parasite withdraws too much nutrient, the invaded cell dies and the mycelium, deprived of nourishment, dies with it. Occasionally the invading mycelium is killed or arrested with little or no mortality of host cells; such host resist-

ance may be either anatomical or physiological (16, 204, 402, 406, 407).

Development of the fungus is influenced by the amount of nutrients absorbed from the host, although there are differences of opinion as to the nutritional levels required for spore production (11, 49, 63, 287, 375). When something happens to reduce vigor of the host, there probably is a reduction in both the number and viability of spores formed. Conversely, incidence of infection and vigor of the rust increases with vigor of the host plants (222, 269).

Sporulation of other fungi in culture has been increased by the use of vitamins and growth-promoting substances (303, 394). With C. fusiforme, manipulation of the host environment and nutrition to increase or decrease the availability of host metabolites and hence to control sporulation has been accomplished with the oak hosts (312, 313) but not with the pines.

Radioactive tracers (389) may be used to evaluate the effect of host nutrition and composition on spore production. They have also been suggested as a means of measuring spore dissemination, but, since very low energy levels may be necessary to avoid injury to the fungus protoplasm, the problems in trapping and identifying tagged spores at any great distance may be insurmountable.

Inorganic nickel salts are absorbed by the host plants, and reduce the incidence of stem rust of wheat (91, 92, 93, 94, 258). There is evidence that these salts adversely affect the respiration of the fungus already established in the host plant (89). Nickel chloride solutions have reduced the incidence of fusiform rust in nursery tests, even when applied several days after inoculation (305).

Metallo-organic complexes, such as ferbam and nickel-amines, are particularly toxic or inhibitory to the rusts (49, 70, 155, 156, 215, 232, 380). Some of these compounds apparently are absorbed by the host plant, thus becoming systemic chemotherapeutants (72). Uptake apparently depends on the cumulative effect of the concentration and frequency of treatment (55, 231, 232). Fungicides also have a pronounced effect on the availability of nutrients to the host plant (70, 253), and indirectly on the parasites. In some cases, the rusts are stimulated by fungicides in very low concentrations (242).

Only a few antibiotics (organic chemicals derived as metabolic byproducts of fungus growth) are known to have any effect on fungi pathogenic to plants. Streptomycin, Terramycin, and cycloheximide are perhaps the most effective (7, 64, 87, 109, 115, 201, 235, 304, 328, 390, 391, 392). Cycloheximide has been relatively successful in reducing rust infections in several species of plants other than pine (8, 115, 203, 236, 391, 401). Its phytotoxicity limits the amount that can be applied, but formulations vary in this respect and the addition of chlorophyllin is reported to reduce the phytotoxic effect (8, 401).

Cycloheximide and its derivatives have a long and controversial history of use against the white pine blister rust (22, 73, 199, 203, 235, 236, 342). Part of its reported effectiveness apparently is due to the fact that, on some of the treated trees, the cankers were parasitized by Tuberculina maxima Rostr. which killed the rust fungus (22, 73, 131, 161, 162, 393). At Gulfport and at the Southeastern Forest Experiment Station, the cycloheximides were found to be phytotoxic to pines at concentrations below which they affected the fungus.

HOST RELATIONSHIPS

Although the most noticeable and damaging effect of fusiform rust is on the pine host, the disease is an interlocking complex of the hosts, the parasite, and the environment. Some part of this complex has been discussed with relation to the physiology of the fungus, and this section will consider the effects of the fungus and the environment on the host plants.

Spore Dissemination and Infection

Infection requires the production and dissemination of spores that must reach, germinate, and become established on and in susceptible host tissues. The number of spores in the environment commonly is summarized by the expression "inoculum potential" (74).

Laboratory studies of spore germination give an incomplete picture of the inoculum potential of fusiform rust. In the first place, the intensity factor is in itself a complex of spore source and the physical factors influencing dissemination of infective spores. Of these factors, perhaps the most important are the number of spores produced by the alternate host, the height from which the spores are released, the distance from the source to the host, wind force and direction, the weight of the spore, and the temperature and humidity.

The number of spores reaching a host will double as production at the source is doubled

(357); in general terms, the probability that a spore will travel a given distance is proportional to the number of spores released at the source. As distance from the source increases, the concentration and deposition of spores decreases very rapidly, the decrease in concentration becoming logarithmic (40, 145, 271, 399, 413). As the sporidia of C. fusiforme are produced during periods of high humidity, they are relatively heavy, and the distance to which they are carried by air currents becomes restricted. As humidity decreases they become lighter and may be carried longer distances, but at the same time are exposed longer to the lethal effects of dehydration and sunlight (198, 200, 262, 346, 358). Spore traps (154, 243, 281) have only recently been used in studying the fusiform rust, chiefly because of design inadequacies in older traps and the difficulty of identifying the spores (135, 180). That periodicity of sporidial discharge is related primarily to moisture conditions appears to be well established (309, 310, 346).

The height at which spores are released also influences the distance to which they are carried (145, 220, 405, 413). Spores from high sources generally travel farthest, but those produced near the ground frequently are elevated by successive thermal updrafts and kept aloft by turbulent air (274, 283, 359). Quite insignificant convection currents may be enough to counteract the terminal velocity of falling spores (110, 111, 220).

Attempts have been made to reduce infection in pine nurseries by eradicating nearby oaks (194, 195, 297, 363), but moderate to high percentages of infection have occurred in spite of oak eradication up to 1,500 feet (195, 302, 358). Sporidia of C. ribicola have been carried by wind 10 to 17 miles from their source (343).

The relative numbers and stage of development of telial columns on sample oaks near the nurseries frequently are used by nurserymen as an index of the need for fungicidal spraying. Here also results have been inconsistent, with occasional very high percentages of infection when telial counts were low. It was once thought that telial production in the upper part of the oak crowns is greater than that at 5 or 6 feet above ground, where counts usually are made. However, a recent study showed significantly more telia at the first 6-foot level than at 12 and 18 feet. It is possible that infection of the oaks generally is less severe in the relatively open areas near a nursery, and that more severe infections at considerable distances from the nursery produce enough sporidia to overcome the diluting effect of distance (343, 348, 349, 358).

No effort has been made to reduce the aeciospore load in the environment. To date, most attempts to control fusiform rust appear to have been derived from analogous practices used in controlling or limiting the cereal rusts and the white pine blister rust (332). Since each disease is an individual biological complex, there is no guarantee that procedures which control one disease will be equally effective with another. In the case of fusiform rust, eradication of the oak host, if it were feasible, would be biologically and economically unsound. The oaks are aggressive and persistent plants, and constitute a large and important component of the southern forests. Moreover both the pine and oak hosts must be considered in their capacities as reservoirs of infection. None of the spore forms are known to survive in nature more than a few months after they are produced. There is no evidence that the fungus remains alive in any oak tissue after abscission of the leaves.

The fungus is perennial only in the pine; living mycelia have been found in cankers more than 50 years old. One infected pine may have aeciospores for several years in succession (11, 41, 66), and for the disease to become epidemic on other pines a considerable amount of inoculum must first be formed on the oaks (292). The pines produce tremendous volumes of aeciospores; one small pine with multiple stem and branch galls probably has more infective spores than do several oaks. An average gall releases from 1 to 4 g. of aeciospores; it is calculated that there are approximately 240 million spores per gram (216). If we assume the lower yield of 1 g. per gall, 10 percent dissemination and deposition on oak leaves, and only 10 percent viability of these spores, we still have two million potential infective aeciospores from each gall. From one to three or more telia on oak are derived from each aeciospore or urediospore infection. Probably the host requires more than one spore, and possibly many spores in an infection court, for the disease to become established (171, 251, 349, 358, 402, 407, 410). Refined inoculation techniques will be required to elucidate both this point and the mechanism of hyphal penetration (148, 193, 250). Existing techniques (135, 171, 278, 279) are applicable only to "mass action" effects where an excess of spores is deposited on host plants to insure infection. Lamb and Sleeth calculated that the average infected water oak tree bears 223,000 telia (195). If, as discussed

above, 10 percent of the deposited aeciospores germinate and establish a mycelium, more than 10 oak trees could be infected at the same intensity by the aeciospores from one gall. Furthermore, the aeciospores are less sensitive to adverse temperature and humidity, and apparently require less time for germination and establishment than do sporidia (290). Obviously, the pine is the main reservoir of infection, and it is logical to assume that control of the disease would be most effective if concentrated on the aecial stage.

The mechanism of infection on the oaks requires further investigation. In nature deposition of aeciospores on the oak leaf may be equal on both upper and lower surfaces (112), but light intensity and humidity differences may inhibit germination on the upper surfaces (48, 77, 84, 284). In C. quercuum, the infection cycle can be broken if the aeciospores are subjected to 2 or more days of adverse environmental conditions (241). While the germ tube penetrates the oak leaf chiefly through the stomata (82), artificial inoculation of the upper surface of oak leaves has occasionally resulted in infection (135).

The time needed for germination and establishment of the parasite in the hosts has been inadequately studied. Infections from aeciospores are reported to require 5 to 11 days for establishment; those from urediospores, 9 to 11 days (135, 290). Infection of pines, expressed as yellow-green (135) or purple spots on infected leaves, requires from 4 to 10 weeks (171). These, however, are gross observations, and the fungus probably becomes established in shorter periods of time. When sporidial germ tubes enter the needle of the pine, establishment of the rust in stem tissue becomes less likely with increasing distance from the stem (265).

In the oaks, C. fusiforme seems to exist only in leaf tissue. On rare occasions telia and uredia have been found on tender stem tissue after artificial inoculations (83), but the fungus has not been found in woody tissues of either naturally or artificially infected oaks. The haploid mycelium in the pine, on the other hand, grows through the leaf tissues and ramifies into the woody tissue (166, 168, 176, 178, 265).

Susceptibility and Resistance

The relative susceptibility or resistance of a host plant results only partly from variations in mechanical barriers to infection, such as the relative thickness of cuticles and the frequency of stomata and epidermal hairs. For the most part, susceptibility depends upon the physiological interrelationship between fungus and host. The host may lack nutrients essential for growth of the fungus, or the pathogen may be stimulated by the composition of the host tissues (119, 164, 222, 354, 407). Toxins may be produced by either host or fungus (29, 38, 204, 406).

For the oak hosts, the term "susceptibility" has been used very loosely. Sometimes it denotes susceptibility to invasion by the fungus, but more often it is used to express the capacity of the fungus to produce telia and sporidia.

From the literature, it is impossible to tabulate the relative susceptibility of species of oaks. Different criteria have been used by different investigators: the frequency with which trees of individual species were found to bear uredia and telia, the number of telia per leaf, the number of telia per unit area of leaf surface, and the number of telia per tree (135, 194, 195, 302). In some cases, failures of artificial inoculation on known natural hosts (135) may have been erroneously interpreted as a reversal of relative susceptibility.

Fifteen species and varieties of oaks are common natural hosts of C. fusiforme. In addition, 30 other species of Castanea, Castanopsis, Lithocarpus, and Quercus have been successfully inoculated with aeciospores and urediospores (132, 134, 135, 194). The known hosts are listed here; an asterisk precedes the name of each common natural host. The names in brackets are the current epithets for the species; the identity of the two Castanea hybrids has not been determined.

Castanea dentata (Marsh.) Borkh.
Castanea mollissima Blume
Castanea pumila Mill.
Castanea sativa Mill.
Castanea henryi × mollissima (?)
Castanea alnifolia × mollissima (?)
Castanopsis chrysophylla (Doug.) A. DC.
Castanopsis diversifolia (Kurz) King
Lithocarpus densiflora [=L. densiflorus (Hook. & Arn.) Rehd.]
Quercus agrifolia Née
*Q. alba L.
Q. bicolor Willd.
Q. borealis Michx. f. [=Q. rubra L.]
Q. californica [=Q. kelloggii Newb.]
Q. cerris L.
*Q. cinerea Michx. [=Q. incana Bartr.]
*Q. coccinea Muenchh.
Q. dentata [=Q. virginiana var. dentata

(Chapm.) Chapm. ex Sarg. = Q. virginiana Mill.]
Q. digitata (Marsh.) Sudw. [=Q. falcata Michx.]
Q. douglasii Hook & Arn.
Q. emoryi Torr.
Q. frainetto Ten.
Q. gambelii Nutt.
*Q. imbricaria Michx.
Q. kelloggii Newb.
*Q. laevis Walt.
*Q. laurifolia Michx.
Q. lobata Née
Q. macrocarpa Michx.
*Q. marilandica Muenchh.
Q. michauxii Nutt.
Q. montana Willd. [=Q. prinus L.]
Q. myrtifolia Willd.
*Q. nigra L.
Q. palustris Muenchh.
*Q. phellos L.
Q. prinus L.
Q. robur L.
*Q. rubra L. [p.p.] [= Q. falcata var. falcata Michx.]
*Q. shumardii Buckl.
*Q. stellata Wangenh.
*Q. stellata var. margaretta (Ashe) Sarg.
*Q. velutina Lam.
*Q. virginiana Mill.
Q. virginiana var. fusiformis (Small) Sarg.

Most references in the literature state that the black oaks are more susceptible than the white oaks (104, 135, 137, 194, 195, 298, 299, 302, 351, 363). If only presence or absence of infection is considered, an analysis of the tabular data in Hedgcock and Siggers (135) shows that the white and black oaks are approximately equal in susceptibility, the weighted infection percentages of the two groups differing by less than 5 percent. If infective capacity is considered, however, the black oaks as a group consistently bear more telia than do the white oaks (20, 194, 195).

The authorities also state that water oak (Quercus nigra) is the most susceptible of the oaks (135, 298, 299, 351), although Lamb (194) considered the blackjack oak (Q. marilandica) and other "scrub oak" species (Q. rubra, Q. cinerea, etc.) to be most susceptible. Here again, the relative susceptibilities depend on the criteria used to evaluate them. The 1938 field notes of Lamb and Sleeth, the basis for the earliest publications on fusiform rust (194, 195), show considerable variation in the occurrence of telia on any oak species, depending on location and date of examination. On the average, Q. marilandica bore 28.9 telia per leaf for all leaves examined, against 14.9 telia per leaf for Q. nigra. When these data were converted to number of telia per average tree, Q. nigra had 223,000 telia and Q. marilandica only 65,000. The dense epidermal hairs on the lower surface of Q. marilandica leaves probably limit aeciospore infections.

There is somewhat more agreement on the relative susceptibility of the pines, as expressed by the formation of galls. Loblolly pine, and then slash and pond pine, are most frequently infected (298, 363). Longleaf pine has a practical degree of resistance; on similar sites, infection of longleaf pine would be only about one-tenth that of slash pine. In the writer's observation, Sonderegger pine (Pinus × sondereggeri H. H. Chapm.), a natural hybrid of longleaf × loblolly pine, apparently is as susceptible as loblolly pine, but it has not been included in any previously published list of susceptible species. The known hosts, native and exotic, are listed here (135, 170, 268, 282, 302):

Pinus canariensis C. Smith
P. caribaea Morelet
P. contorta Dougl.
P. cooperi var. ornelasi Martinez [=P. lutea var. ornelasi Martinez?]
P. coulteri D. Don
P. elliottii var. densa Little and Dorman
P. elliottii var. elliottii Engelm.
P. halepensis Mill.
P. jeffreyi Grev. & Balf.
P. mugo Turra
P. muricata D. Don
P. nigra Arnold
P. nigra var. calabrica Schneider
P. palustris Mill.
P. pinea L.
P. ponderosa [=P. ponderosa var. ponderosa Laws.]
P. ponderosa var. scopulorum Engelm.
P. pseudostrobus Lindl.
P. radiata D. Don
P. rigida Mill.
P. rigida var. serotina [= P. serotina Michx.]
P. sabiniana Dougl.
P. × sondereggeri H. H. Chapm.
P. sylvestris L.
P. taeda L.

P. thunbergii Parl. [= P. thunbergiana Franco]
P. torreyana Parry.

Shortleaf pine and its hybrids have been considered immune to fusiform rust, although susceptible to eastern gall rust (135, 140, 169, 171, 174, 363). Nevertheless, a high percentage of slash × shortleaf hybrids, and some shortleaf × loblolly hybrids, have been artificially inoculated with fusiform rust (172). Some longleaf × slash hybrids also show significant resistance, apparently the effect of the longleaf parent (69).

Several southern pines are completely resistant, among them **Pinus echinata** Mill., **P. virginiana** Mill., **P. glabra** Walt., and **P. clausa** Vasey. In morphological characteristics and chemical composition of their terpenes (234) these four species are quite similar, being grouped in the same section (**Banksia**) in Pilger's scheme of classification (79). Whether chemical composition is responsible for their resistance is, however, a speculative matter.

Wakeley (364) measured incidence of fusiform rust in some 30-year-old longleaf pines whose seedling histories he had preserved. At ages 6 to 8 years some of these pines had appeared resistant to the brown-spot needle blight (**Scirrhia acicola** (Dearn.) Siggers), and these trees eventually developed more rust galls than those that had been infected by brown spot. The phenomenon is unexplained but may be the result of introgression with loblolly pine, which is relatively resistant to brown spot.

Variations in susceptibility of the pines may be a genetic factor not only between species but also between races. Genetic variation in resistance to fusiform rust among progeny of individual trees has been reported for slash pine (20) and loblolly pine (186). When loblolly pines from widely separated seed sources were planted on the same sites they differed significantly in percentage of fusiform rust infection (1, 2, 53, 54, 141, 142, 362, 363, 385). The consistently higher percentage of infection among trees from the same seed sources appeared to be a clear indication of differences in genetic susceptibility to fusiform rust. There is evidence that natural selection operates to weed out susceptible strains and individuals in the pines, the survivors and their progenies representing new, resistant strains (124, 175). The gradual replacement or dilution of susceptible populations with resistant strains or hybrids will reduce the proportion of infected trees in our forests (259).

Anatomy and Morphology

Cronartium fusiforme apparently has very little effect on the oak host (135, 351). Except for the rare occurrence of uredia and telia on tender stem tissue (83), the fungus is restricted to the leaves. In the leaves, the mycelium derived from any one infection is limited in extent, and does not enter or cross the vascular system (82, 222, 269). Externally, growth and sporulation of the fungus ruptures the lower epidermis and causes a limited area of necrosis on the upper epidermis, directly opposite the uredium or telium. By late summer the invaded cells of the leaf die, and a "shot-hole" effect occurs if infection was scattered; with heavy infections, the entire leaf usually dies (135).

Detailed morphological studies of infection have been made by Jackson and Parker (166) in loblolly pine and by Jewell et al. (176) in slash pine. The anatomical changes leading to gall formation are similar in both species. As with several other rusts (50, 149, 177, 191, 333), the presence of the fungus stimulates both hypertrophy and hyperplasia in the cambial initials, resulting in an increase in the size and number of various cells. The most noticeable effect, and the one primarily responsible for gall formation, is the greatly increased number of ray cells, accompanied by a slight increase in the size of these cells. Tracheids, on the other hand, are somewhat smaller than in normal wood. Summerwood is not formed in the annual rings of the gall.

The mycelium is intercellular throughout the gall tissue, except in the pith (166), and generally follows the wood rays. Mycelium and haustoria may, however, be found in the pith if infection occurred when the pith parenchyma was being laid down (176). Growth of the hyphae between cell walls is mainly mechanical, but may be facilitated by chemical (enzymatic) degradation of the wall and middle lamellae (407). Haustoria develop from branches of the intercellular hyphae, which are constricted where they pass through the cell walls. After penetrating the cell walls, they expand to the normal size of the hyphae, becoming 20 to 30 μ long. The haustoria penetrate the parenchymatous cells of the cortex, the phloem rays, and the resin ducts. They do not enter tracheids, although occasionally numerous short branches of the hyphae enter the tracheids through bordered pits (166). Jewell et al. (176) report occasional atypical haustoria in the tracheids of slash pine galls.

The parasite is unable to influence the anatomy of the host beyond the invaded tissues (81, 128, 167, 176, 374), but Jackson and Parker (166) report the production of periderm barriers immediately in front of the advancing hyphae in the phloem. It is postulated that the rust has a hormonal influence (34, 35, 164, 166), for infected branches often survive and grow vigorously, even overtaking the terminal, while uninfected branches in the same whorl die. Infected branches also tend to break dormancy earlier than uninfected tissues. The metabolism of the host tissue appears to be stimulated by the pathogen, infected host cell, or both, and the ability to increase the metabolic rate of host cells may be a factor in successful parasitism (407).

A similar mechanism probably stimulates production of the numerous basal branches that frequently arise from the galls (31, 42, 134, 139, 298). Basal branches, without stem swelling, have not been considered infallible evidence of infection (138, 139, 298), but a very high percentage of seedlings with visible galls have one or more adventitious branches arising from the gall itself. These branches are quite distinct; they are closely appressed to the stem, and have pale green primary needles. A high content of indoleacetic acid has been reported for "witches'-broom" diseases caused by some rusts (35, 62, 108, 214, 273), and this growth-promoting substance may be responsible for the formation of basal branches on trees infected by **C. fusiforme.**

In nurseries large seedlings are more frequently infected than small ones. The hormones produced by the rust may stimulate growth of the seedling. However, Sleeth (302) comments that galls develop slowly on unthrifty and undersized stock; by implication, the reverse is also true, i.e., gall development is proportional to the size and vigor of the seedlings (222, 269). On the other hand, since seedling development usually is proportional to age, the larger seedlings may have developed from the earliest germinated seed in the nursery and thus may have become infected soonest. Percentage of infection is higher with early than with late nursery sowing and germination (190, 297, 298, 302), presumably because temperature, humidity, and spore load become less favorable to infection as spring advances.

On nursery stock, galls usually become visible in 6 months (95) to 8 months (171) after infection occurs. The intensity of inoculum (251) influences the size of the gall and its rate of growth. If the plants are subjected to a heavy spore load, there is a measurable localized increase in stem diameter in 2 months or less. In some instances, a high percentage of seedlings have no visible infection at lifting time, but develop galls during the first year after planted (17, 60, 295, 297, 299, 336, 360, 363). The reason for such latent infection is not known. It has been suggested that gall development is almost imperceptible on small seedlings (301), but the writer's experience is that latent infections are equally common in large seedlings.

The length of time required for the development of the galls on stems and branches of large trees has not been determined. It is probable that here, too, there are immediate and latent responses.

The growth rate of galls has been inadequately studied. Growth in diameter (301) and length (39, 120, 298) apparently is correlated with vigor of the host. Galls develop more slowly on larger trees than on seedlings, and growth rate differs among host species (298). The effect of the fungus on diameter growth of the gall reaches a maximum about 6 to 8 months after infection, or about the end of the first growing season. Approximately the same number of abnormal host cells are produced on the gall each growing season, since the percentage increase in cross-sectional areas becomes progressively smaller as stems and branches increase in size, and with each succeeding year of growth (176).

Branch galls elongate at a rate of 2 to 5 inches per year, and usually grow slightly faster toward the stem than away from it (298). Trunk galls grow down the stem faster than upward (298), and only rarely does upward growth exceed the downward growth (288). The growth of stem galls is somewhat faster on slash than on loblolly pine, but branch galls increase in total length faster on loblolly pine than on slash pine (298).

Typically, galls have a reasonably symmetrical fusiform shape tapering in both directions. Many are hemispherical at either or both ends, and may resemble galls of **C. quercuum**, but these hemispherical galls are not consistent and may begin tapered growth in 1 or 2 years. Occasionally a fusiform gall ceases growing in length and becomes cerebroid.

Branch galls more than 15 inches from the stem tend to be self-limiting, since the parasite generally kills the branch before the hyphae can reach the stem (120, 298, 351, 352). There is a progressive increase in branch mortality with age

of infections. In the first 1 or 2 years after galls become visible, 10 to 15 percent of the branches with galls are killed; by the third year mortality may be 30 to 35 percent. A gall closer than about 3 inches from the stem indicates that the fungus probably will enter the stem within 1 year, if it is not already there (298). On the other hand, Jewell (168) states that the fungus does not grow beyond the swelling, and has not reached the stem even if the proximal portion of the gall is within ½ inch of the stem. Jackson and Parker (166) also report that the fungus does not occur in branch wood or bark beyond the externally visible margin of the gall. However, because the galls usually are fusiform in shape, with a gradual taper, there is no clear-cut line of demarcation between infected and normal tissues. Other criteria of fungus invasion are needed to supplement the gross morphological changes and delimit the extent of infection.

Host Physiology

The physiological processes of the host plants are influenced by the parasite through all stages of infection, beginning with germination of the spore. Great increases in the respiratory rate of wheat occur immediately after rust spores germinate, and the metabolism of host plants frequently is stimulated by infection (61, 63). The physiologic disturbances leading to proliferation of host tissue already have been discussed briefly, and a few other effects may be mentioned.

In studying the Woodgate rust, Hutchinson (164) found slight changes in host cell composition and structure in resistant pines, similar to the periderm barriers reported by Jackson and Parker (166). These changes occurred immediately after invasion by the rust, preventing further growth of the fungus.

Since death of the host cell precedes the death of the fungus, there is a possibility that host cells are killed by toxins or staling products produced by the parasite, as well as by the imbalance between nutrition of the host cells and that of the fungus (29, 38, 204, 406). It has been reported that benzimidazole or similar compounds regulate the formation of wound periderms in white pines resistant to blister rust (35).

Death of individual cells, then groups of cells, in the cambial region creates extensive open wounds, and the gall technically becomes a canker. Fusiform rust sometimes produces cankers without first forming galls (256). Cankers are fairly common on large trees. Siggers[1] followed a logging operation in Alabama and reported cankers up to 23 feet in length, with a rough correlation between canker length and age.

Four percent of the cankers were more than 50 years old, and 22 percent more than 40 years old. While cankers are spectacular, most of the mortality caused by fusiform rust occurs in seedlings and young trees within the first 5 to 7 years of age; only rarely are cankers formed on these trees, and almost never on seedlings. Death is not due to destruction of tissues per se, but to some systemic cause: toxins (29, 214, 406, 407), or interference with nutrition or water transport (49, 210, 269). Death of infected seedlings, or of individual infected branches, is relatively sudden. Seedlings may be exceptionally vigorous, with the foliage normal in appearance for some time; then there is a rapid development of chlorosis, followed by death. These changes often occur within 2 or 3 weeks, thus eliminating the possibility that death is caused by nutritional effects.

Several abnormal metabolic byproducts have been found in the host cells, produced either by the host or the fungus. Excessive quantities of some organic acids, starch, tannins, and gums are reported (34, 38, 119, 166). A copious flow of pitch from galls and cankers is fairly common (135, 398), even on young seedlings in the nursery. The pitch canker fungus (**Fusarium lateritium** f. pini Hepting) sometimes infects trees at fusiform rust galls, and is said to be the cause of this flow (23). However, because the fusiform rust increases the number of resin ducts (166, 176), it probably is often the sole cause of pitch flow.

Ecology and Phenology

Any change in the character of the host population affects the host-parasite relationship (21), and the severity of fusiform rust is definitely influenced by stand composition, age, and density.

A frequent explanation for the rise in incidence of fusiform rust is that control of forest fires has modified the stand composition of the southern forests by increasing the proportion of oaks (337). In the South the pine type is a fire subclimax, and with protection from fire gradually develops into the oak-hickory association. However, widespread control of fire has been in effect for not more than 20 or 30 years, and probably has not influenced the ecological succession much in this time. A more reasonable explanation for the gradual increase of oaks is that pines make up 60 percent or more of the cut, although the forest inventory is predominantly hardwood (337). In other words, the

[1] Siggers, P.V. A study of the development of old rust cankers in a stand of merchantable loblolly pine trees. Unpublished report. Bur. Plant Ind., Soils and Agr. Eng., 7 p. 1943.

proportion of hardwoods (including oak) is increasing because much more pine is being cut.

Another factor contributing to high rust incidence is the increased planting of the most susceptible species, slash pine (298, 299, 351, 363). Although forest managers are concerned about the losses sustained by this species (3), there is no indication that they will replace it with less susceptible pines in the near future. Most of the acreage in the zone of high rust hazard is definitely off-site for shortleaf pine, but some slash-shortleaf hybrids show promise. They have a high degree of resistance to rust, and their silviculture and management apparently will be similar to those of pure slash pine.

Longleaf pine is in disfavor as a planting species, and most of the former longleaf sites are now planted to slash and loblolly pine (299, 300, 363). In spite of its high quality and resistance to fire and fusiform rust, longleaf pine is a difficult species to plant, relatively slow-growing, and subject to considerable mortality and growth reduction from brown-spot needle blight.

The extensive planting program of recent years (338, 339) also contributes to the increased incidence of fusiform rust, because it raises the proportion of very susceptible 1- to 5-year age classes in the forests. Young pines elongate their buds considerably earlier than older ones, weeks earlier in some cases (363), and remain succulent and susceptible for longer periods of time.

The forest stand structure and composition further influence the severity of infection through their effect on the microclimate (100, 158, 358). The amount of ground cover, the density of the overstory, and the presence of windbreaks all influence the temperature and humidity near the ground, and possibly contribute more to the infection of young trees than does the age of the plant per se. In dense vegetative cover, the relative humidity may be at or near the saturation point at the ground level and 30 to 40 percent lower at the 6 foot level (27), which may account for the high proportion of 1- and 2-year-old seedlings which become infected.

Infection of host plants may be limited or reduced by any physical barriers which intercept infective spores and prevent their reaching the susceptible host (74, 111, 283, 324, 358). A mixed forest composed of susceptible and resistant species theoretically should have a lower percentage of infection in the susceptible species than in a pure stand of this species. The same mechanism should also operate where susceptibility varies with age (298, 312, 363); the older, less susceptible plants will intercept some spores and protect the younger, more susceptible individuals. However, Hebb (130) studied pine stands with and without overstories of older trees, and found no difference in the percentage of trees infected with fusiform rust; 28 percent of trees without an overstory were infected, and 30 percent in stands with an overstory.

It has been observed that the percentage of infected trees is lower in dense natural stands than in relatively open stands (205, 207, 223, 237, 298, 363). In part, this is due to the trapping of sporidia, and in part to the more frequent natural pruning of infected branches in dense stands.

Insects, birds, and animals exert little if any effect as vectors of fusiform rust. Flies, wasps, bees, and other insects are attracted to the pycnial ooze and may aid in diploidization of the fungus.

The effect of site quality on the incidence of fusiform rust is not fully known (299). In Alabama, Goggans found that infections increased as the rate of height growth increased, the site quality index being significantly correlated with the number of infections (103, 104). Lindgren stated that vigorous trees appeared to be more susceptible to infection than trees with slower growth rates (207), but Barber (18) found a negligible relationship between height growth and intensity of infection. Dell and Driver (68) reported no correlation of rust incidence with diameter of host trees in plantations.

In general, any cultural practice that stimulates the growth of either host will increase rust infection. Early attempts to improve the growth rate of pine plantations by cultivation and fertilization were found to cause a substantial increase in fusiform rust (17, 30, 102, 103, 104, 209, 298, 351, 363, 388). The explanation is phenological. The cultured trees break dormancy earlier than other pines, have tissues in a succulent stage for longer periods of time, and develop larger areas of succulent tissues.

Winter and spring fires have similar effects. Fire destroys the older, hardened needles, induces an early break in dormancy, and stimulates the production of succulent and susceptible needle tissue (293, 294, 363). Fires destroy some branch galls, but the predominant effect of winter and spring fires is to aggravate the disease.

Weather has a very important influence on both the hosts and the fungus. Climatic differences, more than any other factor, dictate the regional differences in intensity of infection. The pheno-

logical cycle of both oak and pine hosts follows a normal pattern dictated by temperature, precipitation, elevation, and latitude (25, 152, 153). The variable factors are temperature and precipitation, and relatively minor variations are enough to change the dates of phenological development of the host plants and produce significant effects on the fungus (150, 291, 292).

In the Gulf States, February temperatures a few degrees above normal usually induce an early break in dormancy of both oaks and pine. If this break follows a winter mild enough to favor sporulation of the aecial stage of fusiform rust, infection of the oaks becomes very severe, as does subsequent infection of the pines (292). Conversely, very cold winters and springs delay the development of both fungus and host, resulting in very low incidence of infection (302, 355, 358).

A short-range fluctuation in aeciospore production, associated with low temperatures, has been observed for several years. If temperatures remain below 13° C. for 2 or more days in the spring, aecia fail to develop for several days after that. Humid or rainy weather associated with subnormal temperatures favors the growth of molds on the aeciospores and drastically reduces their viability. An analysis of the temperature and precipitation patterns required for sporulation and spore germination showed that sporidial dissemination is governed chiefly by high relative humidity for about 9 hours (307, 309, 310). The production and germination of C. quercuum sporidia require a minimum of 13 hours of 100 percent humidity following a measurable rainfall (241).

Analysis of weather patterns shows that an inflow of maritime tropical air from the Caribbean area into the Gulf States produces a combination of temperature, rainfall, and humidity favorable for basidiospore production and severe fusiform rust infection (65). Relatively minor differences in temperature and humidity, as affected by plant cover and topography, have been correlated to the development of C. ribicola and the incidence of white pine blister rust (272, 343, 345, 346). Gross regional weather patterns as they influence the development of fungi and incidence of other plant diseases have been investigated in detail, and have been used to forecast the severity of these diseases (280, 357, 371, 372).

CONTROL OF THE DISEASE

Fusiform rust has many characteristics of an epidemic disease (or more exactly, an epiphytotic disease) in that increasingly large numbers of the population become, and remain, infected. It is difficult to analyze as an epidemic disease, however, because its development in the forest occurs in successive steps, often several years apart, rather than in a regular, geometric pattern.

Even though the fungus is relatively restricted in the tissues of the pine host, the rust has many characteristics of a systemic disease (347). Generally, a systemic pathogen is safe within the host, and if the host is perennial and the disease not immediately lethal, a long life of the host guarantees a long life to the pathogen (348). The disease can be eradicated only by destroying the host. An exception to this statement may be made where systemic chemicals are effective against the pathogen (64, 72, 160, 183, 184, 240, 326, 349, 391).

Where values are sufficiently high, as in nurseries and seed orchards, fungicides give some protection (45, 78, 315, 382, 383). But the source of inoculum must be reduced or eliminated; otherwise, repeated spraying will be necessary in perpetuity, with no assurance that the seedlings may not become infected as soon as they are outplanted. Nursery spraying is only a temporary delay of infection, not control of the disease.

Considering the range and importance of fusiform rust, the inertia exhibited with respect to its control is surprising. Annual losses are estimated to be over $10,600,000 in sawtimber and 97 million cubic feet of growing stock (3, 264, 337), but expenditures for direct control totaled only $9,500 in 1952, the only year for which cost figures were reported. All of this money probably was spent for spraying in nurseries; it is doubtful that any control was attempted in plantations. Yet seedlings are produced for planting on inadequately stocked sites, and preventing losses in these new plantations becomes a matter of major economic importance.

Control in Nurseries

Shortly after the disease was first recognized in the nurseries (194, 299, 302, 329), trials and experiments were conducted and recommendations for control were published (195, 302). Surprisingly enough, there has been little change in these recommendations in 30 years, the differences being in refinement of techniques rather than in basic principles. The early recommendations called for spraying with bordeaux mixture or ferbam at least twice a week, from the beginning of seed germination until mid-June (297, 302).

Ferbam (ferric dimethyl dithiocarbamate) is the most frequently used fungicide, primarily because the spray mixture is easy to prepare and is noncorrosive. As a fungicide it is generally considered at least as efficient as bordeaux mixture (297), though sporidia of C. fusiforme have been observed to germinate while in contact with clumps of ferbam particles (188).

On the basis of small-scale tests, other chemicals appear approximately equal to ferbam in effectiveness (297, 298, 305). Among these are ziram (zinc dimethyl dithiocarbamate), zineb (zinc ethylene bis dithiocarbamate), puratized spray (phenyl mercury triethanol ammonium lactate), and nickel chloride (183). The high effectiveness of nickel chloride against fusiform rust spores in vitro suggests that this compound should be a good fungicidal spray.

Systemic chemicals have had limited trials, but appeared ineffective (302). Antibiotics (phytoactin, cycloheximide) have been unsuccessful as nursery sprays, mainly because of their phytotoxicity.

Current recommendations are to spray twice a week (and more frequently if rain washes off the fungicide) at the rate of 2 pounds of ferbam and 1 pint of spreader-sticker in 75 gallons of water per acre. Sprayer pressure should be at least 300 pounds per square inch. To insure maximum coverage and effectiveness (44), the spray is broken into fine droplets by being forced through No. 2 nozzles, i.e., having an opening no larger than 1/32 inch (0.031 inch) (96). Since the nozzle openings are eroded by the ferbam suspension, they should be frequently checked, and replaced when worn. Mist blowers have also been successfully used; the application rate is 2 pounds of ferbam in 25 to 30 gallons of water per acre (96, 97, 263).

Some of the spreader-stickers used with protective fungicides are phytotoxic, or cause injury to pine seedlings when used at rates higher than 1 pint per acre (308, 341).

Siggers (290) concluded that abundant infection of pines requires a minimum of 18 hours of humidity close to the moisture saturation point, with temperatures between 60 and 79° F., and suggested that control could be secured if spraying were done when these conditions prevailed (290, 297). In areas of high infection hazard, however, results of such "forecast" spraying have been very disappointing. Extensive trials at two nurseries showed that infection percentages were much higher than with routine spraying. In one nursery, test beds sprayed on the basis of weather forecasts had almost as much infection (19 percent) as unsprayed control beds (21 percent).[3]

It has been difficult to account for 20 to 30 percent infection in nursery seedbeds when hygrothermograph records showed that "critical" conditions did not occur. Some of this high infection rate undoubtedly is due to the fact that temperature and humidity in the seedbeds may be more favorable for infection, and may persist for longer periods of time, than in the part of the nursery where the hygrothermograph is located (27, 100).

There is also some confusion between rain and relative humidity. Some authors recommend spraying before rain, on the assumption that rain equals 100 percent relative humidity (95, 305). However, rain washes spores out of the air and off the hosts (11, 111, 112, 113, 145, 146, 220, 283, 343, 399), thus severely restricting infection. Rain also reduces the protective coating of fungicide, and may remove it almost completely if it has not had time to dry (42, 43, 270, 384, 395). A more reasonable recommendation is to spray as soon as possible after rain, especially if the rain had been heavy enough to wash off some of the fungicide.

Recommendations for late sowing are practical and realistic (195, 297, 302); infection in nurseries was reduced by half with every 2 weeks' delay in sowing date. The effect of sowing date is related to weather conditions. Seedlings resulting from early sowing emerge when humidities are high and temperatures remain below 70° F. for considerable lengths of time; late-sown seedlings, on the other hand, begin growth when temperatures frequently exceed 80° F. (the lethal temperature for sporidia) for several hours a day over long periods of time.

The final attempt on the part of the nurseryman to furnish disease-free seedlings is by inspection and culling of infected seedlings at lifting time (363). This practice is not entirely successful. At most nurseries, shipping schedules demand that unskilled laborers examine seedlings at the rate of 30 or 40 per minute, decide on grades, and determine if the seedling is free of disease. The work is too fast to be effective. Seedlings passed by the graders often include about 5 percent with visible rust symptoms. Moreover, in many nurseries a substantial percentage of seedlings have latent

[3] Unpublished data on file at Gulfport, Mississippi, field office of the Southern Forest Experiment Station, USDA Forest Service.

infections, in which the symptoms are not detectable even by careful examination, but develop in the first season after outplanting (32, 60, 295, 297, 299, 301, 302, 336, 360, 363). Latent infections are roughly proportional to the percentage of visible infections but apparently decrease if the lifting date is late, probably because of the continuing development of the galls.

Control in Plantations and Natural Stands

Possibly the first step in controlling fusiform rust in plantations is to plant only rust-free seedlings. In studies where infected seedlings were planted, mortality averaged 71 percent in the first growing season, and 83 percent of the planted seedlings were dead the second year. Less than 1 percent of the trees survived 10 years after planting (289, 301).

Further control of the disease in plantations is primarily through silvicultural measures. Longleaf and shortleaf pines should be planted on their respective sites whenever possible, and plantations of loblolly or slash pine should not be established where susceptible oaks are abundant, especially if native pines on and near the site have numerous galls (195, 298, 363). If a choice of species is possible, preference should be given to loblolly pine (318, 321). When plantings are made on high-hazard sites close spacings are recommended to compensate for future losses and to provide for earlier natural pruning (78, 144, 223, 237, 298, 363). Infections in dense stands tend to form branch galls. The infected branches die very early, thus reducing the possibility of the more damaging stem galls and cankers.

The selection and planting of resistant races or hybrids of loblolly and slash pine is recommended also (20, 26, 78, 124, 140, 174, 363). The adverse effect of intensive culture (fertilization and cultivation) has been mentioned.

Once the disease is established in a plantation or natural stand, practical control measures are almost nonexistent (298, 363). To some extent, future stem infections can be prevented by pruning infected branches (100, 208, 296, 298, 350, 351, 352). Pruning is most effective in 2- to 5-year-old plantations. It is not economically feasible, however, except on individual high-value trees, and even then reduction in numbers of stem galls has been disappointing in some localities. It is possible that pruning stimulates adventitious sprouts which later become infected. The pruning wounds may also allow other diseases to enter the trunks (403). It is recommended that efforts be concentrated on pruning branches with galls approximately 3 to 15 inches from the stem; galls closer than 3 inches may have already infected the stem, and those farther than 15 inches will die before reaching the stem (120, 208, 350). But leaving any active branch galls will also leave a source of aeciospores in the environment (207).

In timber stand improvement work, treating oaks with chemicals, rather than simply cutting them, will prevent sprouting (194, 363) and thus reduce the population of the alternate host in a stand.

Removing infected pine trees by thinning or salvage cuttings is recommended (187, 205, 206, 207, 299, 351, 363, 386), although it is also suggested that as many trees as possible be left for future salvage cuttings, and to avoid excessive opening of the stand. Wenger (386) states that a cankered tree is safe to leave at least 5 years if any sound callus can be seen extending past the canker along both edges when the marker faces the canker. Lindgren (207) proposed at least three categories:

a. If less than 50 percent of the trunk circumference is killed (cankered), there is more than an even chance of salvage for 8 years.

b. If 50 percent or more of the circumference is killed, but there is no bend in the trunk and no deeply sunken canker face, there is at least an even chance of salvage for 5 years, somewhat less for 8 years.

c. If more than 50 percent of the circumference is killed, and there is a bend at the canker or the canker face is deeply sunken, there is less than an even chance of salvage in 5 years.

The question of whether to remove all stem-cankered trees, or to leave as many as possible, should be re-evaluated from a pathologist's point of view. There is no doubt but that removal of many sporulating galls and cankers will drastically reduce the amount of inoculum (74, 357). This measure was first proposed in 1937 by Lamb (194). Lindgren (207) raised objections to it, mostly from the standpoint of forest management but also because he doubted whether less than a complete eradication program would effect any reduction of inoculum. Wakeley (363) states that even a light initial production of aeciospores to infect the oaks is likely to result in heavy infection

of pines as a result of subsequent secondary infections.

Small-scale trials of excising galls on stems, and using fungicidal paint or antibiotic sprays on the wounds, have been unsuccessful in eliminating the fungus on individual trees. Surgical procedures may be practical when the value of such trees is high (67). It is reported that the application of sodium arsenite solutions to galls is highly successful in killing the fungus, without phytotoxic effects on the pines (37).

Although biological control has possibilities with other plant diseases, it does not hold any foreseeable promise for fusiform rust (192, 323, 408). Six species of pine rusts, including C. quercuum, are parasitized by the fungus Tuberculina maxima, which grows on the pycnia and aecia but is of no importance in control (131, 161, 162, 393) and has not been reported as a hyperparasite of C. fusiforme. Sporobolomyces sp. and Fusidium sp. overgrow the telia of fusiform rust, and Trichoderma, Hormodendrum, and Penicillium spp. are frequently found on the pycnial exudate in the field and in the greenhouse, but the presence of these fungi does not reduce subsequent aecial sporulation of the parasitized galls.

From this review, we must conclude that direct control of fusiform rust in our forests is impossible in the light of present knowledge. A very considerable amount of research still is required on such fundamentals as the life history and physiology of the fungus, and the host-parasite relationship. Only with more facts will it be possible to design a realistic program for control of the disease.

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